

Amendments to the Specification

Please replace the paragraph beginning at page 20, line 14, with the following rewritten paragraph:

The variant HuCC49V10 carries the L-CDR-1 and L-CDR2 of the human antibody LEN, and a threonine at position 97 in the CC49 L-CDR3 is replaced with a serine residue present at the corresponding position in the human antibody LEN. The variant HuCC49V10 also has several substitutions in the heavy chain. Specifically, an asparagine at position 60 in the murine CC49 H-CDR2 is replaced with a serine, a glutamic acid at position 61 in the murine CC49 H-CDR2 is replaced with a glutamine, an arginine at position 62 in the murine CC49 H-CDR2 is replaced with a lysine, and a lysine at position 64 in the murine CC49 H-CDR2 is replaced with a glutamine. It should be noted that HuCC49V10 is described in US. Patent Application No. 09/830,748 (now U.S. Patent No. 6,818,749) and PCT Publication No. WO 00/26394, both of which are incorporated herein by reference. HuCC49V10 was deposited with ATCC American Type Culture Collection, 10801 University Boulevard, Manassas, VA 20110-2209, on August 28, 2003, and has ATCC Accession No. PTA-5416. For the purposes of this disclosure, HuCC49V10 can be referred to as the parental antibody.

Please replace the paragraph bridging pages 30 and 31 with the following rewritten paragraph:

In vitro sera reactivity studies reveal that compared to HuCC49, V10 shows a dramatic decrease in its reactivity to the anti-V region Abs present in the sera of patients who were administered ¹⁷⁷Lu-labeled mCC49 in a phase I clinical trial (Gonzales *et al.*, *J Immunol Methods* 268:197-210, 2002; Tamura *et al.*, *J Immunol* 164:1432-1441, 2000). Using a surface plasmon resonance (SPR)-based competition assay to measure sera reactivity, it has been shown that compared to HuCC49, V10 displays 10- to 300- fold lower reactivity to the sera of several patients (Gonzales *et al.*, *J Immunol Methods* 268:197-210, 2002). Although SDR grafting of mCC49 has rendered V10 minimally reactive to sera from patients who had earlier been

administered mCC49 in clinical trials, the recipient of V10 can still elicit an anti-V region response against the potentially immunogenic murine framework residues that were retained for their presumed indispensability in maintaining the integrity of the Ab combining site (Kashmiri *et al.*, *Hybridoma* 14:461-473, 1995; Tamura *et al.*, *J Immunol* 164:1432-1441, 2000). It should be noted that HuCC49V10 is described in US. Patent Application No. 09/830,748 (now U.S. Patent No. 6,818,749) and PCT Publication No. WO 00/26394, both of which are incorporated herein by reference. These documents also disclose the amino acid sequence of LEN and 21/28'CL. HuCC49V10 was deposited with ATCC/American Type Culture Collection, 10801 University Boulevard, Manassas, VA 20110-2209, and has ATCC Accession No. PTA-5416.

Please replace the paragraph beginning at page 31, line 12, with the following rewritten paragraph:

The antibodies disclosed herein include the CDRs from HuCC49V10 (shown in Table I, below, and in Fig. 9) in a human framework. However, the antibody can also include a non-conservative substitution at position 91 of HuCC49V10 LCDR3 (SEQ ID NO: 11), such as a tyrosine to proline substitution (HuCC49V10-14; see U.S. Patent Application No. 60/393,077, now U.S. Patent No. 6,818,749, and PCT Patent Application No. PCT/US03/20367, filed June 26, 2003, both of which are incorporated herein by reference). Alternatively, the antibody can have a leucine at position 27b of HuCC49V10 LCDR1 (SEQ ID NO: 9) and a non conservative amino acid substitution at position 91 of HuCC49V10 LCDR3 (SEQ ID NO: 11), such as a proline at position 91 (HuCC49V10-15; see U.S. Patent Application No. 60/393,077 and PCT Patent Application No. PCT/US03/20367, filed June 26, 2003, both of which are incorporated herein by reference).

Please replace the paragraph beginning at page 51, line 3, with the following rewritten paragraph:

Synthetic oligonucleotides: Oligonucleotide primers listed below were used for the site-specific mutagenesis of the VL and VH domains of the Ab V10. V10 is described in U.S. Patent Application No. 09/830,748, filed April 30, 2001, (now U.S. Patent No. 6,818,749, herein incorporated by reference). V10 has been deposited with ATCC American Type Culture Collection, 10801 University Boulevard, Manassas, VA 20110-2209, on August 28, 2003 and has ATCC Accession No. PTA-5416. They were supplied by Gene Probe Technologies (Gaithersburg, MD), but could be obtained from a variety of commercial sources. The mutagenic bases are underlined, the positions of the residue changes are parenthetically enclosed, and the sequences recognized by restriction endonucleases are in bold italics.